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Flood-related Outbreak of Peripheral Neuropathy among Prisoners in Bangkok, Thailand

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Abstract

During 9-20 Nov 2011, four prisoners in the national prison died with unknown cause and many prisoners developed diarrhea and neuropathy. The Bureau of Epidemiology launched an investigation to identify etiology and source of outbreak, and implement control measures. A case was defined as a prisoner or guard who developed gastrointestinal (GI) symptoms or neurological symptoms during 1 Sep to 31 Dec 2011. Foods, water, blood and urine samples were tested for heavy metals, vitamins B1 and B12. The prison had 33 guards, 3,668 male prisoners and 555 female prisoners. Among 475 prisoners who met the case definition, 307 (64.6%) GI cases, 49 (10.3%) neurological cases and 119 (25.1%) cases with both GI and neurological symptoms were identified. No case was found among the guards. Attack rates among male and female prisoners were 12.6% (462/3,668) and 2.3% (13/555) respectively. No female prisoners developed neuropathy. Eleven male prisoners had severe distal symmetrical peripheral neuropathy. Four men aged 26-47 years died after developing acute dyspnea. The cases rapidly increased after pipeline of the prison damaged on 2 Nov 2011. Blood and urine samples illustrated vitamin B1 deficiency and high arsenic concentration respectively. This outbreak of peripheral neuropathy possibly resulted from arsenic contaminated drinking water. After providing clean water on 24 Nov 2011, the outbreak subsided within 10 days.

Keywords: peripheral neuropathy, arsenic, prisoners, outbreak, Thailand

Introduction

In Thailand on 22 Nov 2011, Department of Corrections under Ministry of Justice reported to Bureau of Epidemiology (BOE), Ministry of Public Health (MOPH) that four male prisoners from a prison died after developing acute dyspnea, diarrhea, edema of both legs and muscle weakness. Two prisoners died after hospital admission while other two died before they were sent to the hospital. They died 1-4 days after onset of symptoms which were developed following the worst flood in Thailand[1]. BOE, Department of Health from Bangkok Metropolitan Administration, Health Center 43 and Environment and Sanitation Section in Min Buri District joined together and launched an investigation to confirm the outbreak, identify nature and cause of the problem, and implement control measures.

Methods

A cross-sectional study was conducted from 23 Nov 2011 to 25 Jan 2012 in the prison and its surrounding community. For active case finding, we used a structured questionnaire to interview prisoners and villagers about common symptoms, and also performed neurological examination. We conducted door-to-door search to identify community cases.

A prison case was defined as a prisoner or a prison guard who developed at least one of following symptoms: abdominal pain, nausea and vomiting, diarrhea, muscle weakness or sensory loss during 1 Sep to 31 Dec 2011. A severe neurological case was defined as paralysis or loss in sensory function at upper or lower extremities. A community case was defined as a villager living within 500 meters of the prison and had onset of these clinical symptoms during 25 Nov to 10 Dec 2011.

Information of the prison cases was extracted from hospital records of Nopparat Rajathanee Hospital where severely ill prisoners were admitted for care. The extracted information included signs and symptoms, diagnosis, clinical progression and treatment.

For laboratory testing, urine samples were tested for total and inorganic arsenic using standard methods of inductively coupled plasma mass spectrometry (ICP-MS) with high-performance liquid chromatography (HPLC) technique.[2] In addition, blood samples were used to test for cadmium[3] and lead[4] by ICP-MS technique, cholinesterase by enzyme kinetic technique, vitamin B1 by HPLC, and B12 by electrochemiluminescence immunoassay (ECLIA). Vitamin B1 and B12 levels were also tested at the Bangkok Pathology Laboratory. We also requested neurological testing, including nerve conduction studies (NCS) and electromyography (EMG) for two prisoners with the most severe neurological symptoms. Stool and rectal swab samples were tested for enteropathogenic bacteria, including *Clostridium botulinum* and its toxin at the Thai National Institute of Health (NIH). We collected samples among prison cases with non-severe clinical manifestations using convenient sampling method.

Environmental investigation included mapping of prison facilities and interviewing prison guards and prisoners about their activities and exposures prior to illness in order to identify additional risk factors. Water samples were collected and tested for arsenic, cadmium and lead. *Clostridium botulinum* and its toxin were tested in food samples. Cutting boards and food handlers were also tested for enteropathogenic bacteria.

In the community, spot urine and water samples were tested for total arsenic at NIH and Bureau of Occupational and Environmental Diseases.

Results

Description of the Outbreak Site

The prison had a total of 4,223 prisoners (3,668 males and 555 females) and 33 guards. The prison was divided into four zones: 1, 2 and 3 for males, and 4 for females (Figure 1). Prisoners in Zone 1 were waiting for court judgment while wards for long term prisoners were in Zones 2 and 4. Zone 3 was the only zone for vegetables growing. Although male kitchen in Zone 2 served foods to both male and female prisoners, rice was cooked separately in each zone. In female kitchen of Zone 4, food was cooked for prison guards and for sale to prisoners. Due to flooding during 1-15 Nov 2011, the food was changed to preserved food, such as fermented lettuce, fermented bamboo shoots and canned fish.

Pipeline water supply in the prison was connected to the main pipeline of waterworks in Min Buri District outside the prison, with the feeder pipe ran near a police station. The pipeline was divided into two lines: the first line connected to female zone and another line to male zones. Tap water for washing hand, using in bathrooms and watering plants ran in pipes to each building.

For drinking water, tap water in storage tank from Zones 1, 2 and 4 (not shown in the figure) was supplied to water filters in the same zones. However, Zone 3 received drinking water from Zone 2. The filtered drinking water was not sufficient for consumption among male prisoners because the filtering machine in the male Zone 2 had been broken for a long time (Figure 1).

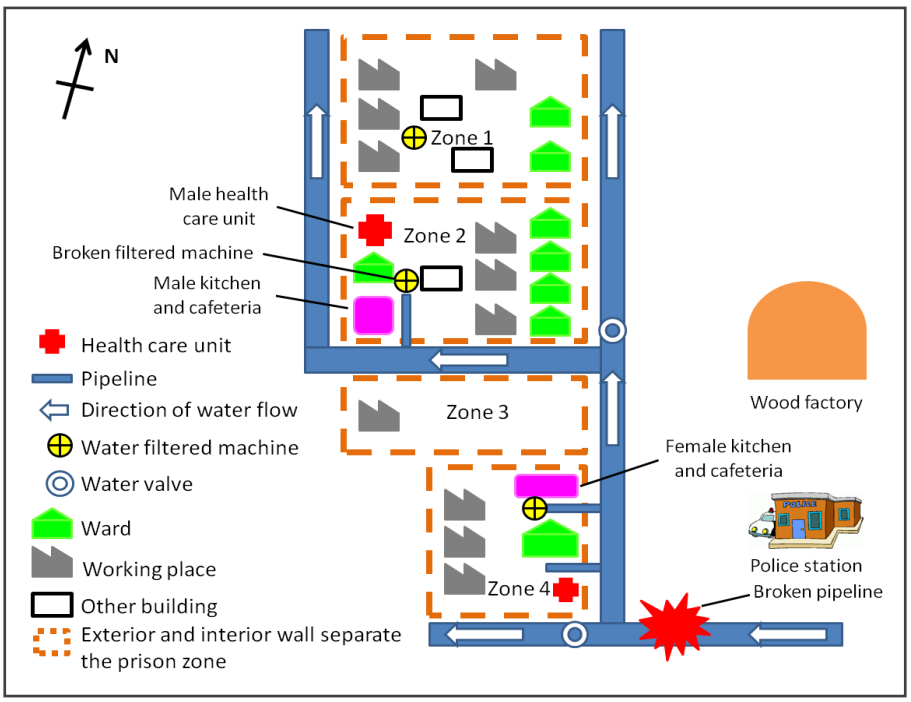

Figure 1. Schematic layout of the prison in Bangkok, Thailand, 2011

Therefore, some male prisoners drank tap water during the flooding. On 2 Nov 2011, the pipeline in front of the police station was broken. After that, some prisoners reported that tap water and drinking water were not clear and oddly smelled.

Outbreak Investigation

Total 464 non-severe cases and 11 severe cases were identified. No case was found among 33 prison guards. Attack rate in male prisoners (12.6%) was higher than that of female prisoners (2.3%). Majority of cases developed gastrointestinal symptoms (Table 1). No female prisoners developed neurological symptoms. Median age on non-severe cases was 30 years (range 17-80 years).

The most common symptoms among prisoners were watery diarrhea (68%), abdominal pain (42%), nausea or vomiting (38%), motor weakness (35%) and numbness (35%). Other symptoms included edema of legs (16%), dysphagia (13%), blurred vision (12%) and ptosis (6%).

There were 460 patients who remembered the first date of their illness which was started from 2 Sep to 3 Dec 2011 (Figure 2). Number of cases increased rapidly after tap water pipeline of the prison was broken on 2 Nov 2011, with the highest on 15 Nov 2011. Then, cases decreased gradually and no more case reported since 4 Dec 2011.

Table 1. Characteristics and symptoms of non-severe cases in the prison, Bangkok, Thailand, 1 Sep to 31 Dec 2011 (n=464)

Characteristic	Gastrointestinal symptom	Neurological symptom	Both symptoms
Total cases	307 (66.2%)	49 (10.6%)	108 (23.3%)
Male: female	294:13	49:0	108:0
Median age in year (range)	29 (17-72)	32 (19-53)	30 (19-80)
Prison ward			
Zone 1 (male, n=536)	0	0	0
Zone 2 (male, n=3,132)	294 (9.4%)	49 (1.6%)	108 (3.5%)
Zone 4 (female, n=555)	13 (2.3%)	0	0
Onset date of first case	28 Oct 2011	2 Sep 2011	15 Sep 2011
Onset date of last case	3 Dec 2011	28 Nov 2011	2 Dec 2011

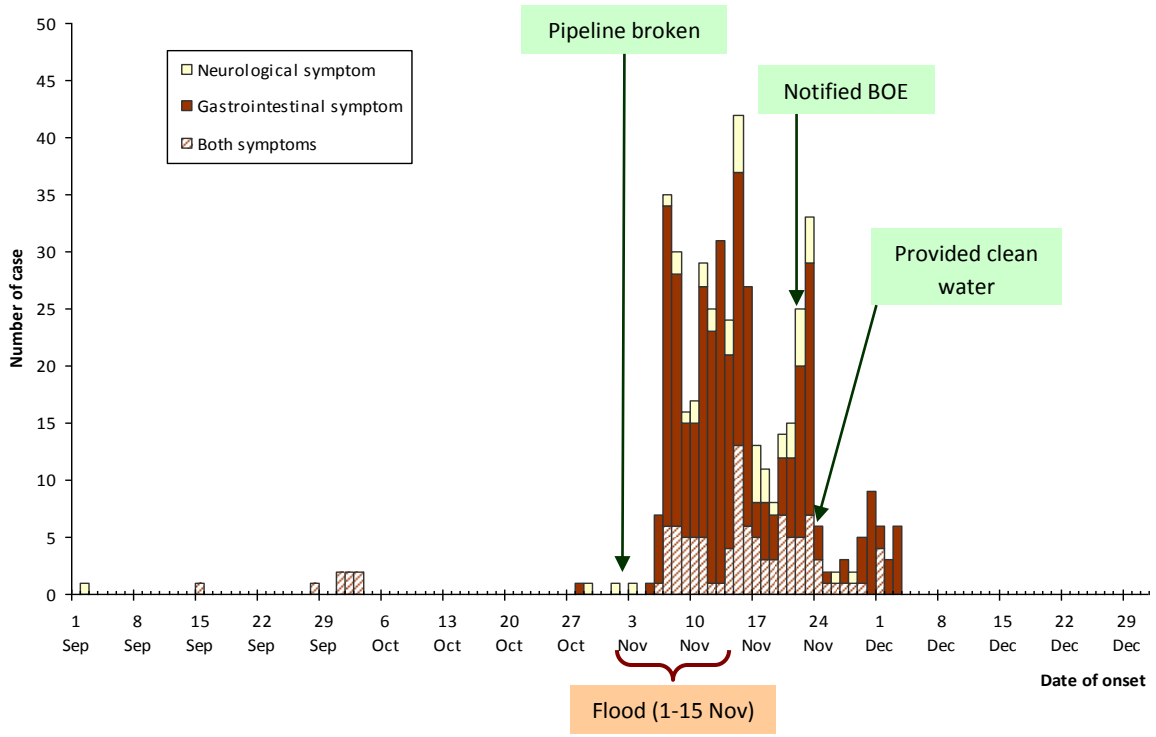

Figure 2. Cases in the prison by date of onset and type of symptoms, Bangkok, Thailand, 1 Sep to 31 Dec 2011 (n=460)

All 11 cases with severe neurological symptoms were males, with median age of 33 years (range 22-53 years). These severe cases were presented with symmetrical distal muscle weakness (Grade III)[5], followed by proximal muscle weakness involving lower extremities only without hemiparesis or eye movement limitation. Among severe neurological cases, 63% of them had sensorimotor deficit while 19% had motor involvement and 18% had sensory deficit. Although numbness was reported, there was no specific pattern of sensory loss. All severe cases had areflexia and also gastrointestinal symptoms. Ten (90.9%) of these severe cases had increased total arsenic concentration in urine (Table 2). Cadmium was also found in two cases (18.2%) and lead in one case (9.1%). All cases had normal concentrations of vitamins B1 and B12. Two cases tested with NCS and EMG presented sensorimotor axonopathy which was not specific for arsenic intoxification.

Information of the firstly notified cluster of four dead prisoners was investigated through other prisoners and guards. They were all males, aged 26-47 years and died after developing acute dyspnea. Their symptoms were ill-defined because of incomplete records. Therefore, we excluded them from the case group.

Laboratory results of ill prisoners and guards showed that 23 out of 54 prisoners (42.6%) and two out of eight guards (25.0%) had high arsenic concentration in their urine (Table 3). Of 24 samples testing for vitamin B1 function, 16 (66.7%) had low function. Among 24 rectal swab samples for enteropathogenic bacteria, *Shigella flexneri* was identified in five samples (25.0%), *Aeromonas spp.* in one sample (5.0%) and *Plesiomonas shigelloides* in one sample (25.0%). None of seven blood and stool samples contained *Clostridium botulinum* or toxin (Table 3).

Table 2. Laboratory results of severe neurological cases in the prison, Bangkok, Thailand, 1 Sep to 31 Dec 2011 (n=11)

Laboratory test	Total	Number positive	Percent
24-hour urine			
Total arsenic ≥ 120 mg	11	10	90.9
Inorganic arsenic ≥ 35 ug/L	11	0	0
Blood			
Cadmium ≥ 5 mg/L	11	2	18.2
Lead ≥ 40 mg/L	11	1	9.1
Cholinesterase > 5,320 unit/L	11	0	0
Vitamin B1 level, aETK* > 1.25	11	0	0
Vitamin B12 level < 211 pg/mL	11	0	0
Nerve conduction studies and electromyography			
Symmetrical peripheral sensorimotor axonopathy involved lower limbs more than upper limbs	2	2	100.0

** aETK - erythrocyte transketolase activity coefficient*

Table 3. Laboratory results of non-severe cases in the prison, Bangkok, Thailand, 1 Sep to 31 Dec 2011

Laboratory test	Total	Number positive	Percent
Spot urine for total arsenic ≥ 50 ug/L			
Male prisoner	41	18	43.9
Female prisoner	13	5	38.5
Prison guard	8	2	25.0
Blood for vitamin B1 activity ≤ 49 ug/L			
Male prisoner	18	12	66.7
Female prisoner	6	4	66.7
Rectal swab for enteropathogenic bacteria			
Shigella flexneri (male)	20	5	25.0
Aeromonas spp. (male)	20	1	5.0
Plesiomonas shigelloides (female)	4	1	25.0
Blood for *Clostridium botulinum* and toxin (male)	4	0	0
Stool for *Clostridium botulinum* and toxin (male)	3	0	0

Table 4. Laboratory results of environmental samples in the prison, Bangkok, Thailand, 1 Sep to 31 Dec 2011

Laboratory test	Total	Number positive	Percent
Filtered water for arsenic, cadmium and lead*			
Zone 2	3	0	0
Zone 4	2	0	0
Tap water for arsenic, cadmium and lead			
Zone 2	2	0	0
Zone 4	1	0	0
Fermented lettuce for *Clostridium botulinum* and toxin	1	0	0
Cutting boards for enteropathogenic bacteria			
Zone 2			
Vibrio fluvialis	3	3	100.0
Aeromonas spp.	3	3	100.0
Escherichia coli	3	2	66.7
Zone 4			
Escherichia coli	2	2	100.0
Food handlers for enteropathogenic bacteria			
Rectal swab			
Plesiomonas shigelloides	12	2	16.7
Hand swab			
Escherichia coli	12	1	8.3

**Reference normal level: arsenic ≤ 0.05 mg/L, cadmium ≤ 0.005 mg/L, lead ≤ 0.05 mg/L*

Environmental Investigation

Environmental samples were collected one week after the flooding. At that time, drinking water and tap water were clear and no odd smell. We did not find heavy metals (arsenic, cadmium and lead) in water samples or *C. botulinum* and its toxin in food (Table 4). *Vibrio fluvialis* (100%), *Aeromonas spp.* (100%) and *Escherichia coli* (80%) were found on food cutting boards. *Plesiomonas shigelloides* (16.7%) and *E. coli* (8.3%) were identified from rectal and hand swabs of food handlers.

We surveyed 31 households in the community (Figure 3). Overall, we interviewed 193 people about history of diarrhea and neuropathy. Of those, we found nine (4.7%) persons with history of diarrhea, but no one with neurological symptoms. Onset of diarrhea was between 15 Oct and 25 Nov 2011. All 31 households drank tap water filtered by a machine or boiled water. The flood affected the community and a wood factory behind the police station.

Of 54 spot urine samples collected from the community, 15 (27.8%) had high total arsenic concentration. However, we did not find any arsenic contamination in seven water samples.

Risk Management

Since the tap water was suspected as the source

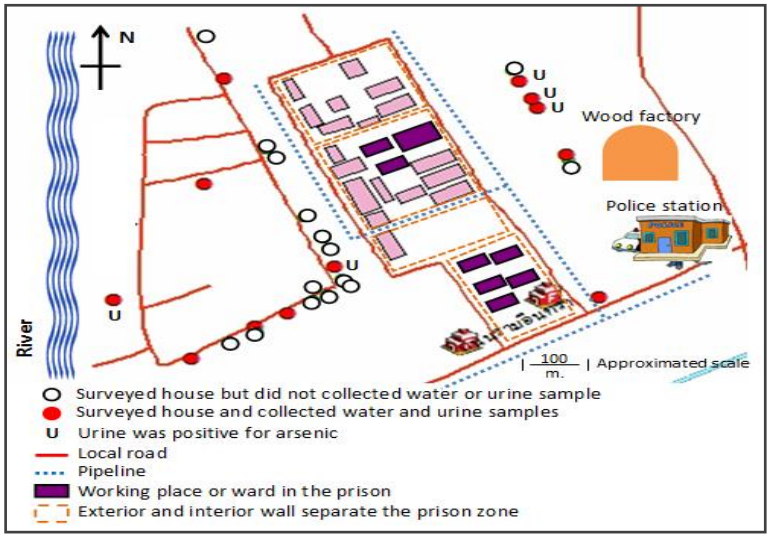

Figure 3. Map of the prison and surrounding community, Bangkok, Thailand, 2011

causing the outbreak, we informed all prisoners on 24 Nov 2011 to stop drinking the tap water and drink only water supply from the Metropolitan Waterworks Authority. Since then, number of new cases rapidly decreased and the outbreak stopped within 10 days. After male prisoners were treated with a combination pill of vitamins B1, B6 and B12 for 2-4 weeks[6], their neurological symptoms were improved.

Discussion

Clinical manifestations and laboratory findings suggested that this outbreak had possibly three etiologies.

One major etiology could be organic arsenic[7-9] due to several reasons. The first one was that arsenic poisoning is associated with nausea, vomiting, abdominal pain and watery diarrhea. Another important clinical sign is peripheral neuropathy which may occur rapidly and is similar to Guillain-Barré syndrome.

For the second reason, laboratory results among cases showed high total arsenic concentration without inorganic arsenic. In general, arsenic substance has two forms of organic and inorganic while the latter can cause more severe health effects. However, high level of organic arsenic can develop health effects as inorganic form. Most tests which measure total arsenic (both organic and inorganic) can give a high value reading when a person eats some fishes or seafood with high organic arsenic. Thus, in order to separate two forms, the ICP-MS technique which measures the level of inorganic form was used in this investigation.

The third reason for neurological cases was that arsenic can cause vitamin B1 (thiamine) deficiency[9-12] by inhibition of pyruvate oxidase that was used in vitamin B1 metabolism. Hence, neurological symptoms similar to dry beriberi were identified in some cases.

In this outbreak, vitamin B1 deficiency could be resulted from not only arsenic intoxication, but also thiamine antagonist[13] found in some fermented vegetables that were eaten during the flooding and malnutrition status because epidemic curve revealed some sporadic neurological cases before the pipeline was broken. These cases might be caused by malnutrition[14,15], or physical or mental stress[16] that can precipitate the neurological symptoms. Although some villagers had toxic level of arsenic in urine, they did not develop neurological signs possibly because they had enough vitamin B1.

For another etiology among gastrointestinal cases, cause of diarrhea could be shigella[17] which was found in rectal swab testing.

The investigation suggested the mechanism of water contamination and the outbreak was most likely to be caused by heavy metals and microbiological pathogens. The wood preservative solution used in the factory commonly composed of arsenic.[18] During the flooding, the wood factory was flooded and chemicals from wood preservatives might have contaminated the flood water. At that time, as the prison pipeline was also broken, tap water could also be contaminated with the flood water. By this way, heavy metals and microbiological pathogens could leak into the pipeline. Coincidently, the water filtering machine in Zone 2, which might have resin to capture chemical contaminants, was also broken and could not filter tap water effectively. Moreover, another precipitating cause could be fermented foods with thiamine antagonist that were eaten during the flood.

Previous arsenic poisoning outbreaks were caused by either intentional or unintentional contamination of food or beverage. A study by Gensheimer et al showed intentional arsenic contamination in coffee.[19] Of 16 people who drank the coffee, 13 (81.3%) vomited and had diarrhea, with one death (7.7%). Another epidemic of arsenic poisoning associated with ingestion of inadvertently contaminated milk powder in western Japan, which resulted more than ten thousand cases and 130 deaths.[20]

Acute arsenic poisoning is associated with gastrointestinal and neurological symptoms even if small amounts of arsenic (less than 5 mg) were consumed.[21] Other symptoms are hematological abnormalities, renal failure, respiratory failure, pulmonary edema, metabolic changes such as acidosis, skin rash, toxic cardiomyopathy and seizure. Lethal dose of arsenic ranges from 100 mg to 300 mg per person.[22]

In general population, prevalence of peripheral neuropathy ranged from 2.4 to 8.0 percent.[23,24] High prevalence may be associated with old age. Diagnosis and treatment of peripheral neuropathy require large amount of time and money as diagnosis often requires special clinical and laboratory tests such as nerve biopsy, EMG and NCS. These tests are helpful to identify pattern of nerve involvement and type of nerve fibers affected, and also narrow down the differential diagnosis.[25]

Epidemiological study for the disease profile in Texas prison[26] showed that major diseases in the prison were communicable diseases such as AIDS/HIV, TB and hepatitis while foodborne outbreaks were also common. Nevertheless, other prisons had reported outbreaks of beriberi due to inadequate food intake.[27-30] A study by Ahoua et al reported that of 5,038 cases recruited, 714 (14.2%) were beriberi cases.[31]

Samples from the four dead prisoners could not be collected for heavy metals testing. However, we hypothesized that the cause of death might be wet beriberi because these cases occurred during the outbreak period and their clinical manifestations were same as severe neurological cases.

Some prisoners could not remember details of illness or risk factors since the outbreak started long time before the investigation. Likewise, heavy metal might already have disappeared from the tap water before the sample collection. Thus, laboratory testing could not detect any heavy metal from the tap water samples.

We could not test the broken water filtering machine whether it was capable to filter microorganisms and heavy metals because the machine was removed from the prison before the investigation. In addition, we could not investigate or test chemicals used in the wood factory and soil around the factory. Finally, there were no more flood water samples available for testing.

Conclusion

This peripheral neuropathy outbreak could be resulted from total arsenic intoxication. Source of the outbreak was probably from drinking contaminated tap water which was carried into the broken water pipeline by the flood water. In addition, foods with thiamine antagonist provided during the flood could be another cause of neuropathy in some cases.

Recommendations

We recommended that maintenance of water supply system in the prison should be improved. The prison should provide adequate amount and types of food to prevent malnutrition and related diseases. A surveillance system should be established in the prison to monitor prisoners with severe illnesses for early detection of future outbreaks.

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